Intraocular pressure during cardiopulmonary bypass

D F P LARKIN, P CONNOLLY, J B MAGNER, A E WOOD, AND P EUSTACE

From the Departments of Ophthalmology, Anaesthesia, and Cardiac Surgery, Mater Misericordiae Hospital, Dublin 7

SUMMARY   Intraocular pressure was measured in 24 patients undergoing elective cardiopulmonary bypass surgery. There was a rapid rise when the bypass circulation began, and this was maintained for about 30 minutes. A simultaneous drop in arterial perfusion pressure and packed cell volume occurred. We suggest that marked haemodilution is responsible for this effect, through increased ocular blood flow and low colloidal osmotic pressure. The possible aetiological significance in relation to ischaemic optic neuropathy complicating cardiopulmonary bypass is discussed.

In this study intraocular pressure (IOP) was recorded with a pneumatonometer at specific times before, during, and after cardiopulmonary bypass (CPB) and the values were correlated with other variables. Despite a lowering of arterial pressure, IOP did not decrease but was increased in all cases.

Two previous studies of IOP during bypass surgery have been performed and conflicting results reported. Stellpflug et al.[1] reported 6–7 mmHg rise in IOP with arterial pressure maintained at 60 mmHg. Lilleaasen and Hørven[2] found that the IOP did not vary significantly from pre-CPB values.

Materials and methods

Twenty-four patients were studied, of average age 55 (range 29–59) years. All were undergoing elective coronary artery bypass graft or valve replacement surgery. None had known eye disease. Informed consent for measurement was obtained from each patient.

Anaesthesia was induced with thiopentone and midazolam. Pancuronium was used for muscle relaxation; anaesthesia was maintained with 50% nitrous oxide-oxygen mixture and intrathecal morphine given. Extracorporeal circulation was maintained with a Stockert non-pulsatile pump and Cobe CML membrane oxygenator. The pump was primed with 2 litres of Hartmann's solution, 25 g mannitol, 60 mEq (mmol) sodium bicarbonate, and 100 mg heparin. This solution is slightly hypertonic (357 mosmol/kg water).

Correspondence to Dr D F P Larkin, Department of Ophthalmology, Mater Misericordiae Hospital, Dublin 7, Ireland.

The IOP was measured with a Digilab pneumatonometer, which is an easy, non-invasive method and gives a permanent record. Calibration was monitored before each case with a Digilab verifier and accuracy checked with Goldmann applanation tonometry at intervals.

Arterial and central venous pressures were monitored via catheters in the radial artery and subclavian vein. Transducers were used for measurement and were calibrated against a mercury column. Blood gas, acid base and haematocrit (packed cell volume) determinations were performed on an ABL-2 instrument (Radiometer, Copenhagen).

Measurements were performed at scheduled times as follows. The IOP was measured in the right and left eyes on several occasions prior to CPB, 5, 10, 15, 25, 45 minutes after the beginning of CPB, and on further occasions after cessation of CPB. Mean values for right and left IOP readings were calculated, and then mean values for the periods before and after CPB were used for analysis.

Blood gas, acid base status, and packed cell volume were measured at the same time as the IOP. Carbon dioxide and oxygen were maintained at normal temperature corrected values throughout the procedure. Body temperature was maintained between 27 and 29°C during CPB.

Continuous values of arterial and central venous pressures were available. Non-pulsatile arterial perfusion pressure was maintained between 45 and 70 mmHg during CPB except at short intervals of deliberate hypotension in 9 patients (see below). The mean central venous pressure before CPB was approximately 7 mmHg. It was not allowed to exceed

this level during bypass, and a variable decrease to below zero was noted in most patients.

Statistical evaluation was by the *t* test applied to a paired comparison.

Results

At the beginning of CPB a significant (p<0·001) fall in packed cell volume was observed. This occurred within 5 minutes of infusion of bypass pump priming fluid. It remained stable during CPB, and increased gradually after CPB. The blood pressure was also significantly reduced during CPB (p<0·001) (Table 1).

The IOP rose immediately in 21 patients, and had risen within 10 minutes of CPB in all patients. The mean elevation for all patients was significant (p<0·001). Gradual return to pre-CPB level was noted at 25 and 45 minutes. A less significant rise was seen at cessation of CPB (p<0·02) (Table 2 and Fig. 1).

The central venous pressure fell to varying levels. No significant disturbances in pH or blood gases occurred.

The increase in IOP correlated with observed haemodilution. A ratio of haemodilution (packed cell volume before/during CPB) and ratio of IOP (highest IOP during/mean IOP before CPB) was calculated for each patient and a graph plotted (Fig. 2). A regression line was plotted from the formula y=a+bx, where y=expected value of y for any value of x, and b=line slope.[3] The correlation coefficient r=0·46.

No complications were associated with measurement procedures.

Table 1 *Arterial pressure and packed cell volume during surgery*

	Arterial pressure (mmHg), mean±standard error	Packed cell volume (%), mean±standard error
Pre-CPB	102·1±2·4	39·0±0·9
During CPB	59·9±1·6	23·7±0·75
Post-CPB	92·1±3·2	

Table 2 *Mean IOP of all patients during surgery*

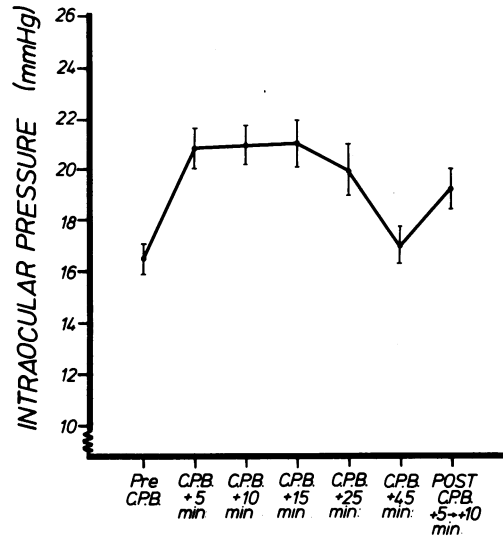| Time	Mean IOP (mmHg)	Standard error
Pre-CPB	16·5	0·53
On CPB 5 min	20·8	0·75
On CPB 10 min	20·9	0·77
On CPB 15 min	21·1	0·84
On CPB 25 min	19·95	0·98
On CPB 45 min	17·1	0·69
Post-CPB 5–10 min	19·25	0·83

Fig. 1 *IOP of all patients (mean and SEM) calculated from mean of each at specified times. As CPB is discontinued in a phased manner, it was not possible to state the precise time post-CPB at which readings were taken.*

Discussion

IOP regulation during CPB is multifactorial, and no clear explanation is evident for the rise observed.

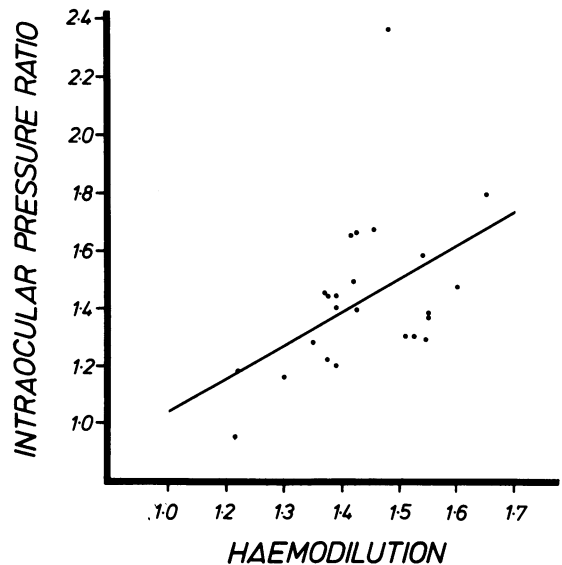Several factors during CPB reduce aqueous production, particularly hypothermia,[4] hyperosmolarity[5]

Fig. 2 *Haemodilution ratio for each patient calculated from packed cell volume before/during CPB. IOP ratio calculated from highest value during/mean before CPB.*

of the infused pump priming fluid, and reduced arterial pressure. Aqueous production is unaffected by reduction of perfusion pressure to 70–90 mmHg, but when reduced to 40–60 mmHg production falls sharply.[6] Presumably this is due to reduced blood flow through the ciliary processes. In nine patients whom we studied perfusion pressure was reduced to 10–15 mmHg for short periods to facilitate surgery, and we noted that the IOP fell immediately to levels around 5 mmHg. Recovery to maintenance levels was immediate also. 25 g mannitol was a component of the pump priming fluid in all patients, and this is probably the cause of IOP reduction within 30 minutes of infusion. Constricted pupils were noted for a period of up to 24 hours after surgery; this is probably an effect of the dose of intrathecal morphine given at the beginning of anaesthesia and would tend to lower the IOP.

Reduced protein content of the blood plasma and low colloidal osmotic pressure increased aqueous production.[7] Haemodilution by the pump priming fluid has this effect, and this partly explains a correlation with rise in IOP. However, the rapid rise in IOP observed would not be fully explained by this non-mechanical cause.

Immediately increased blood flow to the ciliary processes on infusion of pump priming fluid would be a more probable cause. Middle cerebral and internal carotid artery flow velocity has been shown to increase to as much as 300% of pre-CPB levels, and this increase is related to degree of haemodilution.[8] The proportion of aqueous produced by active transport is uncertain, estimates ranging from 35% to 80%.[9] This energy consuming function of the ciliary epithelium requires high oxygen delivery either through increased blood flow, increased blood oxygen content, or decreased affinity of haemoglobin for oxygen. Flow velocity is therefore a critical determinant of the total amount of oxygen released if oxygen content is maintained. Increased flow velocity may compensate for rapid reduction in arterial pressure.

Hormonal changes in CPB may possibly contribute to maintenance of elevated IOP, but would not be responsible for the immediate rise seen. Little systematic experimentation into hormonal effects on the IOP has been performed.

Our findings are similar to those in 50 patients of Stellpflug *et al.*,[1] who showed that a decrease in arterial pressure to 57 mmHg during CPB caused an increase of 6–7 mmHg in IOP. They measured the IOP only 5 and 10 minutes after the start of CPB, and did not specify how the pump was primed. Lilleaasen and Hørven[2] primed the pump with a solution of high osmolality. It is of interest that similar work on pigs showed an increase in IOP during the haemodilution

phase and a parallel increase in intracranial pressure.[10] This shows that tissue fluid content changes are not limited to the eye and raises the possibility of changes in the choroid plexus analogous to those in the ciliary processes. In preference to a Schiøtz tonometer, which was used in the above studies, we used a pneumatonometer. A permanent record was thus obtained and observer error minimised.

Clinical implications of elevated IOP during CPB are not clear. Some elevation or even maintenance of IOP in a setting of arterial pressure reduced to 40–70 mmHg may be especially relevant to posterior ciliary artery supply to the optic disc head. Anterior ischaemic optic neuropathy (AION) involves infarction of the optic nerve head due to compromise of the arterial supply to the laminar and prelaminar regions. Nine cases following CPB have been reported,[11][12] bilateral AION occurring in three; in addition two cases of bilateral AION have occurred in our departments. Sustained elevation of IOP together with reduced ciliary artery perfusion pressure, and a substantial rise in levels of the potent vasoconstrictor angiotensin,[13] are likely causes of AION complicating bypass.

Further study of the behaviour of the IOP during bypass will be of benefit, particularly the influence of pulsatile perfusion, manipulation of priming fluid osmolality, and of mannitol administration 30 minutes prior to haemodilution with pump priming fluid.

References

1 Stellpflug H, Busse H, Niedermeier M, Dittrich H. Augenveränderungen während extrakorporaler Zirkulation. *Ophthalmologica* 1979; **179:** 249–57.
2 Lilleaasen P, Hørven I. Intraocular pressure levels during extracorporeal circulation in man. *Scand J Thorac Cardiovasc Surg* 1982; **16:** 51–3.
3 Bourke GJ, Daly LE, McGilvray JW. *Interpretation and uses of medical statistics.* Oxford: Blackwell, 1985: 131–7.
4 Samuel JR, Beaugie A. Effect of carbon dioxide on the intraocular pressure in man during general anaesthesia. *Br J Ophthalmol* 1974; **58:** 62–7.
5 Davson H, Thomassen TL. The effect of intravenous infusion of hypertonic saline on the intraocular pressure. *Br J Ophthalmol* 1950; **34:** 355–9.
6 Bill A. The effect of changes in arterial blood pressure on rate of formation of aqueous in a primate. *Ophthalmic Res* 1970; **1:** 193–200.
7 Kaskel D, Fink H. Einfluss einiger Kreislauf-grössen auf den Augeninnedrück. *Adv Ophthalmol* 1975; **29:** 190.
8 Lundar T, Lindegaard KF, Froysaker T, Aaslid R, Wiberg J, Nornes H. Cerebral perfusion during cardio-pulmonary bypass. *Ann Thorac Surg* 1985; **40:** 144–50.
9 Cole DF. Ocular fluids. In: Davson H, ed. *The eye.* London: Academic Press, 1984: **1A:** 361–2.
10 Hørven I, Lundar T, Lilleaasen P, Hysing E, Stokke O. The effect of haemodilution on arterial blood pressure, central

venous pressure, intracranial and intraocular pressures in pigs. *Scand J Thorac Cardiovasc Surg* 1981; **15:** 273–7.

11 Sweeney PJ, Breuer AC, Selhorst JB, *et al.* Ischemic optic neuropathy: a complication of cardiopulmonary bypass surgery. *Neurology* 1982; **32:** 560–2.

12 Tomsak RL. Ischemic optic neuropathy associated with retinal embolism. *Am J Ophthalmol* 1985; **99:** 590–2.

13 Taylor KM, Morton JJ, Brannan JJ, Bain WH, Caves PK. The influence of pulsatile flow during cardiopulmonary bypass on the renin angiotensin system and peripheral vascular resistance. *Br J Surg* 1978; **65:** 827.